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Ophthalmoscope as a diagnostic instrument

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THE OPHTHALMOSCOPE AS A
DIAGNOSTIC INSTRUMENT

by

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INTRODUCTION

Ophthalmoscopy permits the study of the interior of the eyeball. It serves not only to detect local intraocular changes in the media and fundus, but also to diagnose remote and general diseases. The importance of ophthalmoscopy can be appreciated when it is realized that the eye is the only organ of the body in which a living nerve can be visualized and studied and the functioning arteries and veins observed and followed to and from their finest ramifications. (29)

Nowhere else in the body can the blood vessels be seen so clearly; nowhere else are they so accessible to direct observation; nowhere else is our duty so imperative to study the minutest detail of the picture as in the vessels of the retina, the more so that they are offshoots from the vessels of the brain, and that certain conclusions can be drawn from their condition with reference to that of the cerebral vessels. Bouchut (1) has termed ophthalmoscopy direct cerebroscopy. This statement overshoots the mark, because the vascular field of the retina can only be considered as a small fraction of a great system, but it indicates how highly

ophthalmoscopy is to be valued. (15)

There is a vast range of disease in which the retina or the optic nerve may show abnormalities, and in many of these, changes in the fundus frequently precede signs of the disease in other parts of the body. In addition, these changes may and frequently do give valuable indications as to the favorable or unfavorable progress and actual prognosis in the specific cases under observation.

This paper is not an attempt to justify the existence of the ophthalmoscope and its use in ocular and general physical examinations, but rather is an attempt to point out the value and necessity of its use as a diagnostic instrument by all practicing physicians, specialists and general practitioners alike.

I. HISTORY OF OPHTHALMOLOGY

The science of ophthalmology is first seen recorded in ancient Babylon-Assyria as far back as 2250 B.C. However, the beginnings of the modern ophthalmology are really found in Greece, where the father of ophthalmology is considered to be Hippocrates - the father of all scientific medicine. He lived in the fifth century B.C., and was the first man to accurately describe the eye anatomically. Though his anatomical description of the eye is still considered as somewhat of an authority, he had entirely the wrong concept of the physiology of the eye. Despite this false concept, he has set down in ancient literature many specific methods of treatment for the various diseases of the eye with which he was familiar.

The next step came in 210 A.D., when Galen came forth with his work on the eye. He also gave a very complete and accurate description of the anatomy of the eye, and substantiated the findings of Hippocrates. However, in addition to this anatomical study, Galen showed where Hippocrates had been wrong in his ideas about the physiology of the eye, and himself wrote up the true correct physiological concept of the eye.

The greatest event in all ophthalmologic history was the invention of the ophthalmoscope by von Helmholtz in 1851. (13) Previous to this time, however,

even in the earliest ages, the eyes of some animals had been observed to be luminous in the dark. Jean Mery of Paris in 1704, held a cat under the water and observed that the pupil dilated, and then he beheld the fundus of the animal's eye. He could not correctly explain the mechanism of this phenomenon, however, but is generally considered to be the first man in modern times to view the interior of the eyeball. Kussmaul, in 1845, removed the cornea and lens of a cat's eye before he was able to view the fundus. Then in the other eye, he removed neither cornea nor lens, but instead removed small quantities of vitreous humor. As the fundus advanced, due to the decrease in volume of its contents, it came within his vision. By this experiment he explained that the fundus lies at the focus of the refractive system of the eye, and that this fact keeps the fundus invisible and the pupil dark under normal conditions.

In 1846 Cuming, in London, performed an experiment in a dark room with a patient, in which the patient sat ten to twelve feet from an artificial light. The subject was asked to look just to one side of the light, and Cuming beheld for the first time the retinal reflex in the human eye. He also noted a silvery white object within the eye, when that eye was moved from side to side. This was undoubtedly the optic nerve head.

The first ophthalmoscope was really invented by an English mathematician, Charles Babbage. (13) However, much to his misfortune, he did not report this invention himself, but turned it over to the foremost ophthalmologist of the time, Thomas Jones, who promptly rejected it as worthless. The following year, in 1851, von Helmholtz invented his instrument, which consisted of a series of three unsilvered plates, laid one upon the other. Since 1851 many men in their work with the instrument have improved it tremendously to make for ease and greater accuracy in eye examination. At first the unsilvered plates were silvered to better reflect light, and a hole placed in the center through which the observer might look into the patient's eye. Finally, Charles H. May improved the illumination greatly by making it solid and free from reflex, shadow, and images of the lamp filament. This instrument of May is the latest form of the original vonHelmholtz ophthalmoscope, and is the one which is at present most widely in use. It consists of a series of concave and convex lenses mounted on a disk which can be rotated in such a manner as to bring one of the various lenses into the aperature through which the examiner observes the eye. By bringing one of these lenses of varying magnifications into place in this aperature, the examiner counteracts the refractive error and accomodative efforts of the patient and himself during his examination of the fundus.

II. THE PRINCIPLES OF OPHTHALMOLOGY

The ophthalmoscope is an instrument consisting of a small mirror which may be plane, concave, or convex, and in which there is a central perforation through which the observer views the subject's eye. The light is reflected by a mirror from an external source or from the source within the handle, and enters the eye of the subject. These rays are then reflected by the retina and the emerging rays pass through the aperture in the mirror to the examiner's eye. (19) In cases of emmetropic eyes, the rays which are reflected by the retina and emerge from the eye are parallel and soon diverge so that they cannot be brought into focus on the retina of the observer. As a result, nothing but a red glow is seen in the normal eye when the patient and examiner are one meter apart. To bring these rays into focus, a biconvex lens must be placed in the path of the rays. However, in abnormal eyes, either myopic or hypermetropic, an image is formed when a distance of one meter exists between the examiner and patient. (2) In the case of myopic patients the image is inverted, while in the hypermetropic patient the image is virtual and erect.

III. METHODS OF OPHTHALMOSCOPIC EXAMINATION

Examination of the fundus of the eye with the ophthalmoscope can be done in an illuminated room without any dilatation of the pupil, but it is greatly facilitated by a darkened room, and rendered more accurate when the pupil is fully dilated. To examine the right eye, the ophthalmoscope is held in the right hand of the examiner, and the right eye of the examiner is used for the examination. To examine the left eye, the ophthalmoscope is held in the left hand and the left eye used.

In all patients there is at first a preliminary examination which should be performed.(19) In this the examiner seats himself one meter from the patient and observes either the red glow from the retina which is observed in the normal eye, or the image of the retina in the cases of abnormal eyes. If there is no red reflex observed coming from the retina, there is some obstruction present within the eye such as an opacity of the cornea or lens, exudates, hemorrhages within the eye, or some other object.

Following this preliminary examination of the eye, one of two methods may be used for the detailed examination of the fundus.

By the so-called indirect method the examiner remains at one meter from the patient and peers through the hole in the mirror of the ophthalmoscope. A

separate biconvex mirror is placed close to the patient's eye, and serves to bring the emergent rays from the eye to a focus between the eye and the eye of the examiner. By moving the lens toward or away from the subject's eye the fundus is brought into focus and clearly seen.

By the so-called direct method the examiner comes quite close to the patient's eye and observes the fundus without the aid of the separate biconvex lens. If the patient's eye is emmetropic, the rays are parallel and are brought to a focus on the retina of the examiner. However, if the eye is hypermetropic, the rays will be divergent and can only be focused by the examiner by the accommodation of his eye, or the interposition of a biconvex lens. If the patient's eye is myopic, then the emergent rays are convergent and a concave lens must be interposed to bring the emergent rays into focus. (6)

This last method, the direct method of ophthalmoscopy, is the simplest and most accurate, and is the one which is most widely used in ophthalmological practice at present.

Using whichever method the examiner prefers following preliminary examination and dilatation of the pupil with some suitable agent, a thorough routine should be followed in inspecting the interior of the eye. It has been said that routine is not a substitute for brains,

but is an aid to our understanding of the patient, This holds true in ophthalmological practice just as in any other phase of medicine. Routine has as much a place in ophthalmological practice as has the ophthalmoscope. (26)

One starts his examination by the use of a plus 20 lens in the ophthalmoscope. With this the cornea, anterior chamber, iris, pupil, and lens are inspected. The magnifying power of the ophthalmoscope is then gradually reduced, and when the plus 16 lens is in place and the ophthalmoscope is two and one half inches from the patient's eye, the beam of light will be in focus and opacities of the cornea, anterior chamber, or lens will appear black like a piece of steel. (6)

To examine the posterior chamber of the eye and the vitreous body, the magnifying power of the lens of the ophthalmoscope is further reduced gradually. The vitreous is usually well illuminated by a plus 8 lens. By having the patient move his eye up and down and from side to side, material in the vitreous may be seen as black particles floating before the eye.

The retina is the next portion of the eye to be examined. By further reduction in the magnifying power of the instrument, the examiner reaches a point of magnification at which the retina is most easily seen. Refractive error in the patient, and accommodation by both the patient and the examiner are counteracted by

the interposition of these lenses so that the fundus can fully and clearly be visualized. Ordinarily, with the patient looking straight ahead and not into the light, the first object noted is the optic nerve head. This should be examined as to size, shape, color, margins, and level with regard to the rest of the retina. If any difficulty is encountered in finding the nerve head, it may readily be found by following the vessels of the retina as they converge.

Vasculature is another of the important parts of the retina which must be studied. The size and caliber of the vessels should be noted, and any abnormalities carefully studied in every detail. Any exudates which are present should be studied as to size, shape, and position, and hemorrhages noted in the same manner.

The last part of the examination is to have the patient look directly into the light, the macula then being seen about one disc diameter to the temporal side of the optic disc. Its color, shape, the presence of exudates or hemorrhages should be carefully noted. (6)

IV. DEVELOPMENTAL ANATOMY OF THE EYE

The eye arises from three different sources in the development of the human embryo. The optic nerve and retina are derivatives of the fore-brain; the lens arises from the ectoderm of the head; and the accessory tunics and the mechanism of accommodation from adjacent mesoderm. (18), (21)

1. The Retina and Optic Nerve:

In the earliest embryos there is seen to be a pair of swellings on the side of the fore-brain. As these swellings grow, they become the optic vesicles which are attached to the brain wall by relatively constricted optic stalks. The continued growth is more rapid on the margins of these vesicles, resulting in an invagination of the distal wall of the vesicle forming the optic cup. This cup is double layered and connected to the diencephalon by a tubular optic stalk, and is destined to become the retina, or the sensory epithelium of the eye. The optic nerve grows from the center of this sensory cup back through the stalk to the brain.

As the optic cup deepens, the cavity of the vesicle is progressively obliterated until the two component layers come to lie in apposition. The outer layer is the thinner of the two and becomes the pigment layer in which pigment granules appear in the cells

and become quite dense. The internal layer is the thicker of the two, and becomes the nervous layer. The limits of this layer become the external and internal limiting membranes. Beneath the external limiting membrane are the layers of rods and cones, which are the receptive elements of the retina. Next, there are several layers of the ganglionic cells, which are bipolar cells and make connections with the layers above and below. Axons from this layer of ganglion cells form the nerve fibers which run to the optic stalk and then grow through the stalk to the brain, forming the optic nerve. The macula lutea is the site on the retina of keenest vision. It is a very small area which lies in the direct optic axis of the eye, and at the bottom of a shallow pit known as the fovea centralis.

2. The Lens:

During the formation of the optic cup, the surface ectoderm overlying the optic vesicle thickens to form a placode which pockets inward to produce the lens vesicle, which occupies the concavity of the cup. This placode detaches from the surface ectoderm, and migrant mesodermal cells fill in the intervening space. The lens vesicle becomes a solid structure, attached by suspensory ligaments to the ciliary muscle, and its cells become transparent so that light rays may pass through the lens to the retina. The lens is a biconvex

structure with a capsule and has the ability to be made more or less convex as the situation requires by pulls exerted by the ciliary muscle upon the capsule.

The space between the lens and the retina becomes filled with a peculiar hyaline fibrillar tissue, known as the vitreous body. This substance is now commonly believed to be a product of the supporting fibers of the retina.

The lens and the optic cup system as a whole become invested with a double layer of condensed mesenchyme. The outer layer of this coat is the more compact of the two and is the forerunner of the fibrous coat of the eye, which later differentiates into the sclera and the cornea. The inner coat is looser and will organize the vascular coat of the eye, including the iris, ciliary body, and choroid.

3. The Choroid:

The choroid coat of the eye is a thin, highly vascular membrane which invests the posterior five sixths of the eyeball. It is pierced by the optic nerve, and at this point is closely adherent to the sclera. It consists of an exceedingly fine capillary network, one of its functions being to provide nutrition for the retina and convey vessels and nerves to the ciliary body and iris. The ciliary body connects the choroid with the peripheral edge of the iris. Part of the

ciliary body are the ciliary processes, which are folds of the choroid which insert themselves between the foldings of the suspensory ligament of the lens. The ciliary muscle consists of unstriated muscle fibers, which are arranged as meridional and circular fibers. This is the chief agent of accommodation. When the muscle contracts it draws forward the ciliary processes, relaxes the suspensory ligament of the lens, and thus allows the lens to become more convex. The iris is a thin contractile

disc suspended between the cornea and lens, and perforated by the pupil. It is continuous with the ciliary body at its periphery. The anterior chamber of the eye lies in front of the iris, and between it and the cornea. The posterior chamber is quite small, and located between the periphery of the iris and the suspensory ligament of the lens. The iris is made up of stroma cells and fibers, and muscular fibers which are both circular and radiating. The circular fibers are the Sphincter pupillae muscle, and the radiating fibers form the Dilator pupillae muscle.

4. The Sclera:

The sclera is the outer coat of the eye, and is a firm, unyielding membrane which maintains the form of the bulb. It is formed by white elastic fibrous tissue, the vessels not being very numerous. The anterior portion of the sclera is the cornea, which is a trans-

parent structure, almost circular in outline, and convex anteriorly. This is a non-vascular structure, but is very rich in nerve supply.

The retina of the eye is supplied with blood by the branch of the Ophthalmic artery, which is a branch of the Internal Carotid artery, called the Central Artery of the Retina. This artery runs within the sheath of the optic nerve for a short distance and then pierces it running the rest of the way into the eyeball through the middle of the nerve, and emerging on the retina from the center of the optic nerve head. The central vein of the retina accompanies this artery.

Other branches of the Ophthalmic artery which supply the eyeball include the short and long posterior ciliary arteries, and the anterior ciliary arteries, which supply the sclera, choroid, and the ciliary body.

V. NORMAL FUNDUS

In order to appreciate the pathological findings of the ocular fundus and recognize the importance of changes in the fundus, one must clearly understand the normal picture of the fundus and the normal variations which may be present. Due to the multiplicity of structures seen in the fundus, no two fundi look exactly alike, and unless normal variations are recognized, mistakes may easily be made. (8)

The so-called optic disc is the picture of the head of the optic nerve, and is quite easily identified by its shape, position, color, and form. It varies in shape from circular to oval, and may be normally quite small or very large. It is usually of a light pink color, paler than the rest of the fundus, and outlined with white or pigmented material forming a distinct arc or ring. The temporal side of the disc is usually somewhat paler than the nasal half. (12) There is a slight depression of the disc in its center which has gradually sloping sides, and which never reaches the edge of the disc normally.

The retinal arteries and veins are seen to course over the disc and entire fundus, and emerge from the center of the optic disc. The arteries are smaller, rounder in form, and lighter in color than the veins. They usually show a very definite light reflex along

their center, a finding which is not present in the veins. (28) The veins, on the other hand are larger and darker in color. The ratio of arteries to veins as to caliber is usually considered to be 2:3. The number of branches and their distribution is never the same in any two patient's eyes, though usually both artery and vein divide on the disc into two superior and two inferior branches. Normally there is no change in caliber of either arteries or veins as they course through the retina, even at the points where they cross. (7)

The macula is the area of the retina in which there is the clearest vision. It is located about two disc diameters to the temporal side of the disc, and is slightly below the horizontal level of the disc. (28) It is seen as a darker area in which there are no visible vessels. There is a bright spot in the center of the macula, which is the light reflex, or foveal reflex, and which moves in the opposite direction to the movement of the ophthalmoscope. The center of the macula may be seen to be yellow in red-free light, but in usual light is simply seen as a lighter red color, with the foveal reflex in its center.

Though this picture of the fundus just described is generally considered as the normal picture, there are numerous variations which are frequently encountered

and which are still considered normal. The veins of the retina may be abnormally large and tortuous, resembling the stasis seen in papilledema. (12) A small artery may be present in some fundi arising from the temporal edge of the disc, and running directly to the macula, the so-called Cilioretinal artery. (8) The thin pigment layer of the retina may show as light colored patches, or the absence of pigment in some areas shows as white patches, the sclera, resembling exudates and retinal degeneration. The macula varies greatly in size and color, and in some may be very dark, while in others there may be no change in color at all from the surrounding retina to mark its position. Some fundi have no foveal reflex because of the shallowness of the fovea, while in others it may be very distinct. (5) In some people, especially children and negroes, a circular reflex may be seen around the macula, with other reflexes radiating from it. This gives a picture which is not uncommonly confused with retinal edema or inflammatory changes.

When these variations in the normal picture of the ocular fundus are recognized and appreciated, the picture presented in the specific case up for examination can be truly evaluated from the ophthalmologic standpoint.

VI. THE FUNDUS IN ARTERIOSCLEROSIS

At the present time the concept of general arteriosclerosis is the result of work done by Gull and Sutton. (20) They believe and have fostered the idea that arteriosclerosis is an independent affection, a general disease of the vascular system. It is not generally considered as a disease in the true sense of the word, but rather comprises changes which may occur with or follow many diseases, the term designating anatomical changes rather than a clinical condition in itself. The clinical features of the condition result mainly from interference with the blood supply of organs or tissues and the resulting disturbance of function. It is defined as a vascular lesion characterized by pathological thickening of the arterial walls, due to a variety of factors, in which inflammatory and degenerative changes as well as hyperplastic and involuntary processes play a large part.

The etiology of this condition is truly unknown, but among the contributing factors are included such conditions as hypertension, endocrine disturbances, overeating, stress and strain, overwork of the muscles, renal disease, and syphilis. It has been said that arteriosclerosis is not a cause of hypertension, although hypertension may co-exist. (83) Cohen believes that hypertension is a secondary, minor, and complicating

factor only. (36)

Arteriosclerosis, as a rule, occurs in the aged, and attacks the larger vessels usually. (37) In cases of generalized arteriosclerosis, sclerosis of the retinal vessels may also be observed. (3) The fundus, however, in arteriosclerosis is often observed to be quite normal. (37) When retinal involvement is present, the lesions are usually mild and rarely progress to the severity of those seen in hypertension. Retinal arteriosclerosis does not often reach the very advanced stages. (28)

R. Marcus Gunn (3) was one of the first observers of the fundus in arteriosclerosis, and set down a list of the retinal findings. However, he believed and stated that these retinal findings were always associated with renal disease.

The lesions of the retina are due to sclerosis of varying degrees of the retinal and choroidal vessels causing hyaline and fatty degeneration of the intima and media of these vessels, encroaching upon the lumen and producing an endarteritis. (36) As to the pathogenesis of this sclerosis of the retinal arteries, it is obviously the same as the pathogenesis of arteriosclerosis in general. Numerous theories have been advanced, but no one knows anything very definite on the subject. Ophthalmoscopic examination lends support to the theory that sclerosis is due to nutritional changes

resulting from vasoconstriction. Gunn believes that constriction of the arteries diminished the rapidity of flow in capillaries and veins with resulting escape of serum into surrounding tissues. Persistence of the edema thus created has been said to interfere with the nutrition of the blood vessels and has led to the occurrence of the sclerosis. (75)

The vessel walls normally in the retina are invisible to the ophthalmoscope, only the column of blood being seen plus the light reflex in the arteries. (8) The normal ratio of artery to vein is about 4:5. As sclerosis occurs, the walls of the vessels become thicker and the blood column narrower in proportion to the veins. The ratio in severe cases may even reach 1:5. This reduction in caliber of the arteries is seen in the entire arterial tree of the retina, though the thickening of the walls is not uniform throughout. There is a definite widening of the light reflex due to an increase in the thickness and the loss of transparency of the arterial walls. Due to the lack of uniformity of involvement of the vessels, the column of blood appears to vary in caliber, and may even take on a beaded appearance in some cases. Usually as the arteries grow narrower they take on the appearance of copper wires. (33)

Agatston (23) states that the process begins with spasm of the arterial walls, which are segmentary con-

tractions of the muscular coat, causing the artery to fade in color at the site of spasm, and which come and go with no definite outline. These spasms result in permanent narrowings at the site of the spasms, which have a definite and constant outline, similar to the neck of an hour-glass. As the process continues, tortuosity of these arteries results, and is considered as a fairly constant finding of retinal arteriosclerosis.

Along with the changes just noted as occurring in the arteries of the retina, changes also occur in the veins of the retina as a result of the arterial changes. One of the most striking and constant signs observed with the ophthalmoscope, is the phenomenon known as Gunn's Crossing Phenomenon. (23) Here, as the artery crosses the vein, the vein narrows or disappears just under the artery. This compression of veins is the result of the thickened arterial wall plus the increased tension in the artery encroaching upon the space of the softer vein. (33) The vein distal to this narrowing becomes distended and tortuous, and also quite engorged. As the result of this compression of the veins by the arteries, they are seen to be alternately broad and narrow, so that they also appear to be beaded. The compression and engorgement of the veins results in a varying degree of venous stasis, which in turn results in the increased permeability of the venous walls.

This increase in the permeability of the walls of the veins gives rise to the passage of elements from the blood stream itself into the surrounding tissue of the retina.

Hemorrhages are also frequently seen along the courses of both veins and arteries. These hemorrhages are either flame-shaped or round, and are sharply circumscribed as a rule, depending upon the depth at which the hemorrhage occurs in the tissue of the retina. (8) These hemorrhages are usually small and not very numerous, though occasionally larger ones occur. The small hemorrhages may absorb completely and leave no scars on the retina, but there are usually signs of retinal atrophy strewn with granular pigment in these areas - scars which are permanent. These scars which are seen with the ophthalmoscope appear as light areas or spots which have highly pigmented borders. (23)

Along with these hemorrhages are frequently observed white plaques of exudate in the retina. These white plaques may be formed by the organization of the hemorrhages, by the deposition of fibrin, or by the lipoid infiltration which occasionally occurs into the areas of the retina which have degenerated due to lack of blood supply. (8) These white patches may also be seen right in the walls of the arteries at times, giving them a lighter color, and having the effect of broadening the

light reflex of the arteries still farther. These are usually the result of exudation of fibrous materials, and may become white cords along the arteries. (28)

The exudative areas are usually white, shiny, flat, hard, and quite irregular in outline, with a definite tendency to coalesce. At times they may appear as very large white areas covering a very sizable part of the retina. (33) As a rule they are permanent findings, and remain in the retina, though occasionally they may be absorbed as the hemorrhages are. (12)

The process of retinal arteriosclerosis is a patchy one and very irregular in occurrence, in which some areas of the retina are markedly affected, while others are not affected at all. The white plaques and hemorrhages are both most frequent about the disc and especially in the macular region. (8) Local vascular accidents are very apt to occur during this process, the most common being the thrombosis of the central vein.

It is generally believed that changes in cerebral vessels as a rule correspond fairly closely with those in the retinal vessels. (8) For this reason the progress of the ocular condition is considered as secondary in importance to the condition of the heart, kidney, and brain, but it is taken as a fair indication of the progress of the general arteriosclerosis in these other

vital organs. (36) Though it is now generally considered that in these cases of retinal arteriosclerosis there is no reason to believe that there is or has been any renal pathology responsible, everyone still believes that when they see a case of retinal arteriosclerosis part of their routine of examination should include careful study of the patient for the presence of renal disease. (3)

Death in arteriosclerosis usually occurs from apoplexy, but may occur from heart or kidney failure because of the widespread involvement of the arterial system of the body. (20) After recognition of marked retinal sclerosis, the patient as a rule will be dead from one of these aforementioned conditions within four years, and not uncommonly death will occur in the matter of a very few months.

This then gives us another clue as to the usefulness of the ophthalmoscope. We can see and watch the progress of the generalized arteriosclerosis in the body as indicated by the changes in the vessels of the retina, and at the same time have a fair idea of the length of time the patient has yet to live.

VII. THE FUNDUS IN HYPERTENSION

In considering the subject of hypertension no attempt will be made to discuss those types of hypertension for which there is a definite known organic etiological basis. The only type which will be considered is that type known as Essential Hypertension, because the findings in the other types are consistent with the findings of the basic pathological condition in each type and will be discussed separately.

Essential Hypertension is a condition in which there is a definite and permanent increase in the blood pressure without any evident cause for this increase. Osler (20) believes that variations in blood pressure do not constitute diseases, but result from disturbances in many and varied diseases. It is generally considered that in order to warrant the diagnosis of hypertension, a patient must have an elevation in blood pressure which is permanent, and the figure of 160 systolic pressure has arbitrarily been taken as the minimum reading.

The real cause of hypertension is not known except in very rare cases. The condition itself leads to changes in the vessels of the body and to renal changes which may terminate in chronic nephritis. There is a markedly increased and persistent high blood pressure with no other disease present to account for its severity. The systolic pressure is the only part

elevated as a rule early in the disease, the diastolic remaining normal or only slightly elevated.

Angiospasm is considered by some as the primary factor in the vessel changes seen in hypertension, and is considered to result in sclerosis of these vessels. (36) The cause of the spasm is thought to be a vasomotor disturbance due to hyperfunction of the adrenal glands, or to a toxic factor similar to that seen in the toxemias of pregnancy. It used to be thought that the retinitis which is seen in cases of hypertension was caused by some toxin which was a nitrogenous waste product accumulating in the blood as a result of kidney damage. Nephritis was always considered as a causative factor, and a complicating factor in hypertension or arteriosclerosis in which these retinal findings were present. (79) Now it is generally known that retinitis seen in hypertension has little or no direct relationship to nitrogenous retention, and that retinitis rarely if ever occurs in cases of urea retention as seen in prostatic obstruction, hydronephrosis, and other kidney disorders.

Most authorities divide hypertension into two completely separate types. The first is the milder of the two and is called simply Essential Hypertension, or Benign Essential Hypertension. The other type is the more severe type and is called the Malignant

Essential Hypertension.

In the benign form of hypertension the patient is usually in perfectly good health for years, showing no symptoms or signs of their condition other than a moderate elevation in their blood pressure. Ophthalmoscopically there is very little noted in the ocular fundus. In many of the cases there is no change in the fundus at all, but many times one sees moderate and localized arteriolar sclerosis and venous engorgement. (37) There is a normal ratio between the arteries and veins as to size. In cases in which there are lesions of the fundus, there is usually slow progression of these lesions, and unless there is adequate control of the condition, this progression usually leads into the more severe malignant form of hypertension. (37)

As there is a progression of these early and mild lesions of the retina, the ratio of the arteries to the veins changes from normal to about 3:3. The retinal arteries and veins become moderately distended and tortuous, and the retinal venules have a corkscrew appearance. (57) A hypertensive arc forms which can be distinguished from the nicking of the vessels which is seen in arteriosclerosis by the apparent narrowing of the vein at the site of crossing without any peripheral venous dilatation.

Adequate control of these patients usually results

in stopping this progression of lesions, and in the actual regression of them. This type of hypertension is not fatal and not severe in itself, but its severity lies in the fact that when uncontrolled it progresses into the more severe and highly fatal form of malignant hypertension.

Traube (72) stated in 1861 that the retinal lesions seen in hypertension occurred only in cases in which there was a hypertrophied heart, especially of the left ventricle. Fishberg and Oppenheimer say that his hypertrophy of the left ventricle is not only present in all of these cases, but that it actually precedes the appearance of the retinal lesions.

In 1876 Gowers (72) pointed out for the first time that in hypertensive states the arteries of the retina were contracted. Since then it has been shown that hypertension is not the result of contraction of the large arteries, but rather the result of contraction of the terminal segments of the vascular tree, the arterioles and capillaries. After contraction or spasm of these small branches has continued for some length of time, permanent changes occur in the walls producing arteriosclerosis. In the capillaries as the result of prolonged contraction, the walls become fatigued and dilate, causing stasis of the blood, and allowing the appearance of elements of the blood outside of the

vascular walls. (72)

Malignant hypertension is considered by some authorities as a separate entity marked by a very acute onset of hypertension, and generally seen in younger individuals. (37) By other authorities it is considered as a simple progression of the benign form into a more severe and fatal form. (57)

In malignant hypertension there is found to be a high diastolic blood pressure, and visual disturbances and fundus lesions appear early. (37) The lesions vary in character and severity. The first sign in the fundus is hyperemia of the disc caused by the venous engorgement which results from the contraction of the arterioles. This hyperemia changes into actual edema of the retina in a short time. (28) This edema of the retina is the most marked around the nerve head, and finally the nerve head itself becomes edematous. The nerve head swells and becomes blurred with indistinct margins, and the fluid and fibrin collects between the radial fibers of the retina, especially about the macula forming a white star-shaped figure. The swelling of the nerve head may become so extreme as to resemble papilledema, and there may be actual retinal detachment in this areas as the result of the transudation of fluid beneath the retina. (8) Visual disturbances may reach the point of complete visual loss.

When hypertension is not complicated by arterio-sclerosis, the first retinal vascular sign is that of generalized contraction of the arteries and arterioles. The ratio between arteries and veins becomes 1:5 or less, and as the contraction continues, thickening of the arterial walls becomes evident along with the indentation of the veins and the dilatation of the vein. (8) These retinal arteries are end arteries, and so as the result of this prolonged contraction and organic change, nutrition of the retina suffers. This gives rise to an infiltration of fatty granular cells forming a variety of brilliant white, sharply defined lesions. (57) In some cases of hypertension, especially in the early stages when retinal lesions are just beginning to form, one may be able to see with the ophthalmoscope the appearance and disappearance of these contractions of the arterioles. These contractions are not permanent, but come and go from time to time.

The veins of the retina are likewise affected, the changes being quite easily identified. They become quite markedly engorged and dilated, the ratio between the arteries and veins having already been mentioned as far from normal. They also become quite tortuous, and arching and compression occurs at the points where they are crossed by the spastic or sclerosed arterioles. (37) Localized retinal thrombosis of any of the branches of

these veins is not an uncommon occurrence.

Hemorrhages, which as a rule occur along the course of the vessels are quite numerous, and are almost always flame-shaped in outline. They are not confined to any one area of the retina in particular, but are fairly generally distributed throughout the retina.

Exudates are also very commonly seen. These are usually more grayish in color than those seen in arteriosclerosis, and are quite ill-defined in outline. The edema in the region of the macula results in the development of a partial or complete star-shaped figure in this region. This figure is made up of white dots with the fovea as its center. (12) This star-shaped figure of the macula is considered one of the most characteristic fundus lesions of hypertension.

In the type of Malignant Hypertension which is considered to be the slow progression from the benign form, certain steps in the development of the retinal lesions are recognized, but do not vary a great deal in character from those already mentioned. The only great difference is that these lesions develop quite slowly, rather than with great suddenness, and there is great difficulty in recognizing when the benign form becomes the malignant form. (57) First there is a change from scattered and localized patches of arteriolar contraction to a uniform contraction of all

of the retinal arterioles, which may become so marked that only the veins are visible on ophthalmoscopic examination. The relation of the arteries to veins changes as before stated, and a well developed and marked hypertensive arc forms gradually at the arterio-venous crossings.

It is generally considered that in hypertension more than in any other disease which produces fundus lesions, the character and progress of the lesions is of very great value in evaluation of the course and prognosis of the general condition. If edema of the nerve head is present, some authorities state emphatically that there is renal involvement which is quite marked. (36) All believe that edema of the disc and surrounding retina usually means serious involvement of the vital organs such as the brain, heart, and kidney, and state that the seriousness of hypertension is based upon the damage done by this condition to the brain, heart, and kidney. (37) Gifford (8) states that patients with a marked nerve head swelling may be expected to die within one year, but most authors give the patient upward to five years before death is expected to ensue.

Wagener (79) states that the presence of sclerosis of the retinal arterioles is definitely diagnostic of primary hypertension, and says that the presence of this sclerosis is an indication of chronicity rather than of activity of the hypertensive disease. He believes

that the persistence of generalized constriction of the arterioles is usually a sign of serious prognostic significance. Angiospastic changes in the retinal arterioles are indicative of activity and progression of the hypertensive disease.

The presence of hemorrhages, edema, and exudation in addition to arteriosclerosis in the retina is considered a more serious and progressive form of the disease, than in those cases where there is sclerosis alone. Wagener (79) states that the retinitis which develops in hypertension is really the formation of infarcts of the retina, and are permanent changes.

Few patients with a marked retinitis live more than three to five years after recognition of the lesions, and some authorities go so far as to say that ninety percent of these patients will be dead before the three year limit is reached. (8)

VIII. THE FUNDUS IN NEPHRITIS

The association of kidney disease with lesions in the eye dates as far back as 1836, when Bright mentioned the fact that visual disturbances might occur in the course of chronic nephritis. Retinal changes were observed anatomically in 1850 by Tuerck, and ophthalmoscopically by Heymann in 1856. However, it was not until 1916 that any distinction was made between arteriosclerotic retinitis and the retinitis of nephritis. (50)

In general it may be said that retinal lesions occur only in those forms of nephritis which are associated with vascular hypertension, or with marked secondary anemia. (20) Retinal changes in Nephrosis are extremely rare, and it has been said that the fundus shows no involvement whatsoever in acute forms of nephritis. (36)

The differentiation of the retinitis of hypertension from that of chronic glomerulonephritis is frequently quite difficult. Cohen (37) believes that when hypertension and arteriosclerosis accompany nephritis, one cannot distinguish the fundus picture from either of these accompanying conditions. Yater's (83) idea on the subject is also that the retinal pictures of hypertension and retinal pictures of renal disease with hypertension are indistinguishable, and then goes on to

state that in all cases of the chronic form of glomerulonephritis and in the cases of late subacute glomerulonephritis, there is a very severe form of hypertension present regularly. From these statements of authorities it seems evident that there can be very little if any difference in the ophthalmoscopic picture seen in nephritis and that seen in hypertension.

The subject of the pathogenesis of the retinal lesions seen in nephritis has been argued for years with no definite conclusion having been reached. Some authorities believe that it is due to nitrogen retention, but this has been fairly well exploded by the absence of retinal lesions in cases of nitrogen retention caused by urinary obstruction in which there is no retinal pathology present. Others believe that there is a hypothetical toxin liberated by the diseased kidney, or that there is a local arteriosclerosis which is responsible, and some ascribe to the theory that there is an ischemia of the retina which causes the appearance of the lesions. (50) Volhard (36) stated quite definitely that in chronic glomerulonephritis there is involvement of the arterioles of the kidney by sclerosis, with the production of a toxic factor which is responsible for the retinal lesions. It is now generally believed that the origin of the lesions is vasospastic and ischemic, because of the frequency with which vasospasm is seen in these cases.

It has been found that retinal observations vary considerably from time to time, depending upon the phase in which the disease is at the time of examination. (50) Retinitis may develop and persist, or may regress and disappear completely. In some cases no retinal pathology will be evident, in which cases the retinitis either developed early and disappeared, or never developed.

In the early stages of nephritis with no hypertension present, the fundus is seen to be quite normal. (28) As the condition develops and progresses, the disc becomes quite pale, and some retinal edema is noted. This edema of the retina is seen to progress in amount and is found to be the greatest on the nasal side of the disc. There is a characteristic lack of the outstanding vascular changes as seen in either arteriosclerosis or hypertension. The vessels appear quite normal, but occasional hemorrhages of the retina may be noted. These are usually flame-shaped and not very frequent in occurrence. In very severe cases of chronic glomerulonephritis without hypertension, one may occasionally see a very mild amount of sclerosing of the retinal vessels, but this is a very late occurrence. (37)

In cases of chronic glomerulonephritis in which there is hypertension present, there is a distinct difference in the picture of the fundus. Here the

picture seems to depend mainly upon the amount of hypertension which exists. If mild hypertension accompanies the nephritis, the picture is much the same as that seen in Benign Essential Hypertension. (37) If the more severe forms of hypertension are accompanying the nephritis, the picture may simulate that of, or may even have superimposed upon it the picture of Malignant Hypertension. (12)

In these cases of nephritis with accompanying hypertension, as in the cases of hypertension alone, the fundus shows first generalized contraction of the retinal arterioles, with eventually sclerosis of these vessels. Also there is edema of the nerve head and surrounding retina, with the formation of a star-shaped figure in the macula, and in the very severe cases detachment of the retina with visual loss. (12) The veins show engorgement, dilatation, tortuosity, and become compressed to a marked extent where the sclerosed arterioles cross them. Hemorrhages, which are rather common in cases of hypertension, or in cases of hypertension accompanying nephritis, are very uncommon in cases of nephritis without accompanying hypertension. They are usually flame-shaped in type. Exudates are present and are indefinite in outline, wooly, and rather a gray or dirty white color. (28)

In a study of 56 cases of chronic glomerulonephritis, made by R. W. Graham, (50) 10 cases were found to have normal fundi until the time that they died. In two

others there had been a few small hemorrhages noted early in the course of the disease, but these had disappeared some time before death. One case showed nothing until just before death when exudates developed, and four others showed nothing until hemorrhages developed just prior to death. The other thirty nine cases showed typical retinal pictures of edema of the retina, cotton wool patches, hemorrhages, and abnormalities in the retinal vessels. In all cases, the onset of the retinitis was considered as acute. The most characteristic picture seen in these 56 cases was an acute angiospastic retinitis without evidence of arteriosclerosis, but in all of these cases with marked retinal pictures, hypertension was an accompanying or complicating factor in the disease.

Once again we find that the ophthalmoscope is a great aid in cases of chronic glomerulonephritis in diagnosing the condition, watching its progress, and as a prognostic indicator. The prognosis of chronic glomerulonephritis is very poor in general, and death is said to usually occur from uremia within three years after the appearance of the fundus lesions. (37) Should malignant hypertension intervene or be present from the start, death usually occurs sooner and is due to either uremia, heart failure, or cerebral accidents. The onset of a retinitis of a purely angiospastic type without any sclerosis of the retinal vessels is an extremely

grave prognostic sign, the mean subsequent duration of life with such retinitis being about four months. (50) This statement is made because of the fact already mentioned that spastic changes in the arterioles indicate activity and progression of the condition, while sclerosis indicates chronicity and lack of activity.

Yater (83) has stated that the development of a neuroretinitis with edema of the disc and retina, and the vascular changes noted already, indicates that renal failure is inevitable. Such a picture never occurs until late in the course of kidney disease, and therefore, is really a sign of impending death.

IX. THE FUNDUS IN DIABETES MELLITUS

Diabetes mellitus is a disease of metabolism, especially of the carbohydrates, in which the normal utilization of carbohydrate is impaired with an increase in the sugar content of the blood and glycosuria. (20) This disease usually occurs in people past the middle age, at the time in life when arteriosclerosis is also almost always present to some extent. (37)

Retinal manifestations of Diabetes mellitus occur as a late manifestation of the disease, and are not considered as very common in occurrence. Diabetes may cause the death of the patient without any appearance of retinitis, or in other cases the picture in the fundus may lead to the diagnosis of the diabetes. (28) Consequently, no person can predict what the picture seen by the ophthalmoscope will be before looking into the fundus, as one can in so many of these diseases which cause a retinitis.

The etiology and pathogenesis of the retinitis due to Diabetes has been discussed for years, with a considerable difference of opinion arising as the various investigators bring out something new as to the basic cause of the retinitis. The result has been that there have been numerous theories with the various groups of authorities aligning themselves with the theory that they as individuals most favor.

Glycosuria was one of the first theories put forth as a basic cause of the retinal lesions seen and is generally considered by most authorities as of some etiological importance, though the actual connection is quite vague. Supporters of this theory believe that the sugar found in the urine in diabetics increases the renal function and irritates the renal epithelia to such an extent that it causes marked renal damage. The retinal changes are said to be due to this renal damage. However, if one analyzes this and assumes that it is a true statement, the retinal changes are then seen to reflect the renal affection primarily, and are only indirectly related to the glycosuria, thereby decreasing the etiological importance of the glycosuria, and making it only one of perhaps many contributing factors. (10)

Another theory supported by some is that the hyperglycemia and hypercholesteremia seen in diabetics is the important factor. However, after investigation, most authors agree that this is entirely theoretical and has no substantial background. Others believe that renal disease is present in diabetes, and is the cause of the retinitis. The arguments advanced by this group of supporters are dispensed with by the majority of authorities by the statement that diabetic retinitis requires no renal component whatever for its appearance. (10)

The theory which is most strongly supported by the

largest number of investigators is that the hypertension and arteriosclerosis seen in diabetics is responsible in some way for the retinal picture. (37) They believe that diabetics are predisposed to hypertension, and since the relationship of diabetes to arteriosclerosis has long been known, that these two factors play a big and very definite part in the etiology of the retinitis. (10) However, there is a fairly general realization that the mechanism of Diabetes is not as yet thoroughly understood, and that until the subject of diabetic metabolism and its effect upon the capillaries has been cleared up, no one will be able to definitely understand the cause of the lesions of the fundus. (20)

Hanum (10) in his monograph on Diabetic Retinitis, has set up a classification of diabetic retinitis, considering that there are four definite and distinct types. The first he calls the Exudative Diabetic Retinitis, and states that this is the type that is most frequently observed. In this type the exudative elements of the retinitis predominate, and are found for the most part in the central part of the fundus. The second type is called the Circinoid Diabetic Retinitis, in which there are exudative elements which have a tendency to coalesce and extend in the form of an arch around the nerve head. The third type is the Hemorrhagic Diabetic Retinitis, in which there is a predominance of hemorrhages, with very

little if any tendency to the formation of exudative plaques. These hemorrhages are present in very large numbers and are not confined to any one area of the fundus as a rule. The last type is the Proliferative Diabetic Retinitis, in which hemorrhages form in the vitreous, and are replaced by fibrous tissue strands growing from the retina into the vitreous.

In a study of 2360 cases by McKee (62), 476 cases were found to have retinal lesions related to their

diabetes. He definitely believed that the most pre-

valent lesion was that of arteriosclerosis of the retinal vessels. The type of sclerosis which was seen in the retinal arteries was that which consisted of a hyaline thickening of the media without complete lumen obliteration. The result of this sclerosis in the fundus was observed to be a general congestion of the veins resulting in an increase in their diameter, and a tortuosity. This congestion results or predisposes to retinal hemorrhages. The effect upon the arteries was found to be just the opposite, with the column of blood decreasing in size and caliber showing great variations. In the very severe cases which were far advanced the walls of the arteries were so thickened that they were actually visible to the examiner.

Gresser and Pillat (10) observed in their investigations that edema of the retina was a fairly common

finding in diabetic retinitis. However, most authorities disagree with this, and some actually state that it never exists in uncomplicated cases of diabetic retinitis.

Hanum considered that the presence of edema of the retina was a most important differential diagnostic point in favor of the retinitis seen with renal disease. Most investigators agree with him, and state that the papilla in diabetic retinitis is likewise unaffected as a rule.

Hemorrhages in the retina were rarely absent in these cases of diabetic retinitis; in fact they were considered to dominate the picture. (10) Even in the first type of retinitis mentioned, in which the exudative elements were said to predominate, there were also noted numerous hemorrhages in the retina of each case examined. McKee (62) disagrees with Hanum, and believes that hemorrhages are the next most common retinal finding to retinal arteriosclerosis, rather than exudates.

Hemorrhages were generally observed to be of two distinct types. The first type were small and round and deeply located in the retina. They were punctiform and spotlike, with both central and peripheral distribution. The second type were flame-shaped or striate in form, with irregular contours. These were more extensive and had a marked tendency to occur in the central areas of the retina. In either type of these hemorrhages they were found to be bilateral as a rule, with only five out of 195 cases having hemorrhages confined to one eye.

In general there was found to be no relationship between the location of the hemorrhages in the retina and the location of the vessels. (10)

Hemorrhages are due to a degeneration of the vascular walls, according to Cohen,(37) and not to hypertension. These changes are permanent pathological vascular changes because though the general condition of the patient improves on dietary and Insulin therapy, there is no improvement in the lesions seen in the fundus. Actually there is an increase in the severity of these lesions.

Though diabetes is generally considered to be a disease of middle age and later, it does occur with considerable frequency in the earlier age groups. It has been proven that in these earlier age groups the disease is much more severe than later in life. In the cases examined of Diabetes in persons between the ages of six and thirty one years, only a very small minority were found to have any lesions of the fundus at all. Those lesions which were found were hemorrhagic areas on the retina.(62) Therefore, an axiom was put forth which seems to be quite timely:- "As Diabetes grown milder with each increasing decade, the hemorrhages become more numerous." (62)

Exudative elements are the other type of lesions seen upon the examination of the fundus of a diabetic. They have for years been considered as the characteristic

ophthalmological picture of diabetes, the argument still raging as to whether these or retinal hemorrhages are the most frequent in occurrence. As with hemorrhages, these elements are also present in different forms in the retina. In the early stages of diabetic retinitis they are small and round and quite discrete. In the later stages there is a confluence of these small early plaques forming areas with polygonal contours, tongue shaped projections, or with deep notches. (10) There is a marked tendency for them to be bilateral, as seen previously in the case of hemorrhages. The plaques present a marked affinity for the central regions of the retina, often forming a more or less complete wreath-like circle around the center of the fundus. (10)

The color of these exudative plaques varies in different cases from brilliant white to a wax-like yellow or a greyish white. Gifford (8) believes that the typical appearance of these plaques is that of small round or oval deposits in the retina which have a yellowish or fatty appearance. Atkinson (3), on the other hand, states that usually these spots are brilliant white and clustered around the macula. In very advanced cases of diabetic retinitis these small areas merge and fill the entire macular region with a fluffy snowbank deposit.

Also noted in the late stages of diabetic retinitis was the tendency for a proliferation of connective tissue of the retina to occur. This proliferation causes a

condition spoken of as Diabetic Retinitis Proliferans.(12) In this condition there are usually fingers or projections over the retina which follow the course of the vessels, are almost white in color, are made irregular in outline by the presence of hemorrhages, and simulate the condition of proliferation found in the retina of syphilis. At least a portion of this proliferation is the result of organization of extravasated blood from the retinal and vitreous hemorrhages. (10)

Since the introduction of Insulin, diabetic coma has largely disappeared as a cause of death in diabetics. However, Insulin has had no effect upon the mortality from cardiac, vascular, or renal disease, these types of lesions being the great cause of death in diabetics now. (62) For this reason, recognition of arteriosclerosis is the only means by which any control can be exerted over cardio-vascular-renal lesions. In spite of very careful general physical examination, sometimes this condition of arteriosclerosis cannot be detected. As a result, ophthalmoscopic examination again comes into the foreground as the main means in these cases by which sclerosis can be recognized. In the fundus one can easily see that sclerosis of the retinal vessels is present, and can judge from its presence and degree the vascular picture in the rest of the body organs. Experience has shown that ophthalmoscopic examination

is not an adjunct to physical examination, but a necessary part of it and should, therefore, be considered as a routine, (62)

X. THE FUNDUS IN INTRACRANIAL LESIONS

Changes in the fundus are very common in various types of intracranial conditions, but are considered as only suggestive and in no way pathognomonic of any of these conditions. The findings are, therefore, of very little value alone, and must be combined with the general manifestations of the condition present to be of any value. In all of these intracranial conditions the signs which may or may not be seen in the fundus of the eye are due mainly to the increase in intracranial pressure created by the underlying pathology. (12)

Probably the most frequently seen and most commonly occurring lesion in the fundus in intracranial disease is that of papilledema. Another name for this finding is choked disc, and it is defined as a non-inflammatory swelling of the optic nerve head resulting from increased intracranial pressure and from the obstruction of the orbital venous outflow. (12) The condition is usually bilateral, but one eye may be affected before the other, or as some authorities have stated, one eye only may be affected. There has been considerable investigation upon the subject of the mechanism of cause of choked disc with numerous theories advanced. However, the exact mechanism is still unknown. The general belief at the present time is that it is due to increased intracranial pressure forcing cerebrospinal fluid into the vaginal space of the

optic nerve causing stasis and compression of the vessels with the resulting venous engorgement. (8)

In the development of papilledema the first change noted is an aggravated redness of the disc. Soon there is edema and swelling of the disc and a progressive increase in its size. The borders of the nerve head are elevated, and the edema spreads to the adjacent portions of the retina. Striations are seen to run from the center of the nerve head onto the retina. The retinal veins become engorged, distorted, and very tortuous, and hemorrhages are observed upon and surrounding the edematous papilla. (3) Early in the development of the choked disc there is no impairment of vision, but later on reduction in vision may and often does occur with complete loss occurring if the intracranial pressure is not relieved. (12)

Brain tumor is the most frequent cause of increased intracranial pressure and choked disc. It is said to occur in at least 80% of these cases, and is very often discovered only upon routine examination with the ophthalmoscope. (28) Hamilton (53) believes that it is a very late sign of increased intracranial pressure. It does occur with great frequency and quite early, though, in Cerebellar, Ventricular, and Temporo-sphenoidal tumors, because these cause an internal hydrocephalus and an early rise in the intracranial pressure. The

size of the tumor is not considered as great a factor in determining the degree of papilledema as the location of the tumor. (67)

The papilledema seen in cases of intracranial tumor is usually bilateral but may be unilateral. (49) The argument has raged for years as to whether unilateral choked disc was of any value in the localization of the tumor. At the present time it is generally felt that it is of no localizing value. Leslie Paton (53) in 1909 was the first to show this definitely. He had quite a heated argument with Victor Horsley in London, and showed on Horsley's own patients that unilateral choked disc was of no aid in locating the tumor.

Another cause of choked disc is brain abscess. This finding is said to be one of the most constant in cases of brain abscess, being demonstrable in from 70-90% of the cases. (3) The changes in the nerve head are the same as those seen in any case of increased intracranial pressure, no matter what the basic cause may be, and so it is generally considered bad practice to try to distinguish one from the other on the basis of the ophthalmoscopic findings alone.

Other causes of choked disc include syphilis which acts generally through intracranial gumma, and tuberculosis which acts through abscesses of the brain and through meningitis. Pituitary tumors as a rule do not cause

choked disc. (12)

As the choked disc is allowed to progress the vision becomes progressively worse, and eventually there is complete and permanent loss of vision. Sachs (67) states that he had never seen a patient who has been blind even a short time as a result of choked disc regain his vision.

Another type of lesion observed in the fundus which runs choked disc a close second in occurrence is that of optic neuritis. There are considered to be two definite types of optic neuritis, the first of which is called the intraocular type, and the second the retrobulbar type. (3)

The intraocular type of optic neuritis is usually present with no symptoms other than a diminution of vision, which is not always present. There is a swelling of the nerve head seen which obliterates the physiological cupping and obscures the outlines of the nerve head. There are also reddish or gray striations which extend from the disc into the retina. These striations along with the swollen nerve head and adjacent retina make the area appear as one large swollen mass. White patches are usually visible at the periphery of the nerve head, and hemorrhages into the retina are not uncommon. The retinal arteries are usually much smaller than normal, while the veins are enormously swollen and engorged and tortuous. Usually both veins and arteries in the region

of the papilla are obscured from view, being buried in the swollen mass of papilla and retina.

The retrobulbar type of optic neuritis may have no intraocular manifestations, or may show the same picture as the intraocular type. Usually, however, the findings in the fundus are not as marked as in the previous type. Almost always there is some edema of the papilla present.

The intraocular type of optic neuritis is the type encountered in cases of intracranial lesions. Either type may and frequently does result in optic atrophy

and loss of vision if allowed to persist.

Many of the common intracranial lesions give this picture of optic neuritis in the fundus, but these lesions are not always seen in all cases, and the absence of a fundus change does not mean a great deal. It is now known that rapidly growing tumors are more liable to be accompanied by optic neuritis than the slowly growing ones. The growth position has no direct influence upon the establishment of the optic neuritis. Likewise, the size of the tumor seems to be of little importance in creating changes which have been noted in the nerve. In cases of brain tumor, the optic neuritis is usually bilateral, though there may be much more marked involvement of one eye than the other. In cases of cysts in which retrogression occurs, or in syphilis as seen in gummas of the brain which are under adequate

treatment, the optic neuritis may clear up entirely and very rapidly leaving no residual.

In differentiating between optic neuritis and choked disc in the fundus, the following points should be kept in mind: (28)

1. Loss of vision occurs early in cases of optic neuritis, but is a late occurrence in cases of papilledema.
2. There are usually no inflammatory signs present around the nerve head in choked disc, while these signs are quite prominent in optic neuritis.
3. The swelling of the nerve head is usually more marked in choked disc cases than in optic neuritis.
4. Secondary optic atrophy may occur in the late stages of both conditions.

Atrophy of the optic nerve is another finding in cases of intracranial lesions. Simple optic atrophy may often be associated with intracranial lesions, tumors especially, but it is said to never occur unless there is some compression of the optic tracts by the mass itself or by ventricular effusion. (3) Simple or primary optic atrophy is a degeneration of the optic nerve fibers without any inflammatory background. (12) Intracranial intradural tumors never cause primary bilateral optic

atrophy. Therefore, if there is evidence of an intracranial lesion and a bilateral primary optic atrophy, the lesions must be extradural, and the only lesions producing such a picture are located around the optic chiasm. These are most frequently pituitary tumors and internal carotid aneurysms. (67)

Secondary optic atrophy is the so-called post-neuritis or inflammatory atrophy. In this type there is also a degeneration of the fibers of the optic nerve, but this degeneration is accompanied by proliferation of connective tissue upon the nerve head. (12) Sachs (67) believes that the appearance of secondary optic atrophy in cases of choked disc is of great prognostic value, because he claims that he has seen several patients with this type of optic atrophy and loss of vision, and in none of these has there been any return of vision. On the other hand, he has seen many cases of primary optic atrophy due to pituitary tumors regain normal vision following removal of the tumor. Other investigators have found the same to be true in a large series of Pituitary and Suprasellar tumors. (12) The explanation offered is that we must be dealing with a physiological block of the nerve in secondary atrophy, and with a simple mechanical block in primary optic atrophy. (67)

By the use of the ophthalmoscope one can distinguish between primary and secondary atrophy. The primary type

has a disc which is white, regular in outline, and sharply defined. On the other hand, the secondary type shows a disc which is white, somewhat hazy, and which has very irregular outlines. In the primary type the retinal vessels and surrounding retina appear to be quite normal. In the secondary type, the retinal arteries are narrow, the veins normal in size but generally quite tortuous, and both the arteries and veins may often be enclosed by white lines. (12)

The ophthalmoscopic signs are of little value in diagnosing thrombosis of Cavernous sinus, because other ocular signs such as lid edema, exophthalmos, muscular paralyses predominate the picture. (80) However, there are usually signs evident in the fundus in addition. There is some venous engorgement as a rule, and a low grade papilledema is the rule. Generalized retinal edema is not an uncommon finding in these cases. Hemorrhages are very infrequent, and so it is said that thrombosis of the central vein is likewise infrequent. To contradict all of these statements, Parsons (80) says that the fundi may and often do remain quite normal throughout the course of the disease.

Bilateral papilledema occurs frequently in thrombosis of the lateral sinuses, and does not necessarily indicate a poor prognosis. Occasionally unilateral papilledema has been seen, but it is generally believed and has been

stated definitely by Dill and Crowe (80) that this is due to involvement of the cavernous sinus along with the lateral sinus. Benedict (80) points out that it is bad practice to try to state definitely the cause of papilledema which is seen other than on a basis of increased intracranial pressure. He believes that choked disc indicates nothing more than a simple increase in intracranial pressure which has existed for some period of time.

Tumors of the meninges fairly often give rise to ophthalmoscopic findings, at least often enough to consider here. Optic neuritis is the most constant finding in the fundus in meningeal tumors. (31) Such a neuritis may be present for months before other indications of meningeal involvement are present. In the very rapidly growing tumors the optic neuritis is said to be quite aggravated compared to the type which is seen in the slowly growing types in which neuritis is not such a marked finding.

Likewise, in meningitis, signs may occur in the fundus. There is usually an optic neuritis in cases of septic meningitis which live long enough for this sign to develop. It is a very severe form of neuritis, and at postmortem examination pus is frequently found within the sheath of the optic nerve. In some cases pus may also be found beneath the retina, and in

children especially, this may resemble a glioma.

Optic neuritis is also present in cases of syphilitic meningitis, as is also the case in tuberculous meningitis, but in neither of these cases is the picture diagnostic enough to be conclusive of the presence of the disease without the other general findings. (12) In tuberculous meningitis white spots are frequently seen on the retina along with typical white streaks which follow the course of the vessels. It is generally thought that the optic neuritis of tuberculous meningitis is of the descending type, travelling along the nerve sheath and nerve itself

from the meninges of the brain to the nerve head and retina in the eye.

The last division of intracranial conditions which produce changes in the ocular fundus to be considered is that of head injuries. In cases of concussion of the brain, it was formerly thought that there were marked changes in the fundus, but at the present time it is generally believed that any changes found are the result of trauma directly to the eye which accompanies the trauma to the head. (3) However, in fractures of the skull, there are apt to be very marked changes noted in the fundus. In cases of depressed fractures or cases which have undepressed fractures but in which there is a blood clot under the site of fracture, there are changes in the nerve head which range from simple edema to marked

optic neuritis. Hemorrhages into the retina, especially around the disc are quite apt to be present in cases of depressed skull fracture. In cases in which there has been marked injury to the meninges accompanying the fracture, there is apt to be a violent optic neuritis which may lead to optic atrophy. The involvement of the optic nerve in the injury to the skull may lead to separation of the vessels of the nerve, and then the picture is the same as that in embolism of the central retinal artery. (12)

XI. THE FUNDUS IN THE TOXEMIAS OF PREGNANCY

The term "toxemia of pregnancy" is one which is very widely and loosely used, but one about which very little is understood. In using the term it is assumed that the blood contains toxins or poisons, but their nature is not understood. These toxins are supposed to be the result of deficient or abnormal metabolism in general, or to be the result of morbid processes in the mother. Another view as to the origin of these toxins is that they are supposed to come from the fetus or the placenta. One can readily see that with so many supposed sources of the toxins, not much is really known about their true source, nor about their true nature for that matter.

One manifestation of toxemias of pregnancy is a change of one type or another which occurs in the eye and can readily be observed by the use of the ophthalmoscope. It has been stated that at least 90% of pregnant women present some ocular symptom at one time or another during their pregnancy. Fortunately, the majority of these are fleeting and inconsequential. The eye has already been observed to be a sensitive indicator of metabolic changes and the presence of toxins in the body. Metabolic changes are normal and an essential part of pregnancy, and in many cases there is a greater or less retention of toxins, especially those resulting from

the metabolic processes peculiar to the pregnant state. (31) In these latter cases toxemia is said to be present.

In the toxic pregnant woman, the slightest disturbance in the eyes is a danger signal and warrants immediate thorough ophthalmoscopic examination. In fact there should be a systematic examination of the eyegrounds in all pregnant women, for this may show up changes which reflect a general process of toxemia which has not yet become evident clinically. (31) By this ophthalmoscopic examination no lesions may be found at all, just as in some cases of all other disease affecting the eyegrounds. However, one may find the same picture as that seen in moderate or severe cases of malignant hypertension. By the discovery of these lesions and the observation of their course, the physician is constantly on guard, and able to head off many of the serious consequences which would have occurred if the eyegrounds had not disclosed the process which was taking place in the body.

The lesions seen in the fundus are due to either the toxemia, the hypertension present, or to complicating nephritis or pyelonephritis. (37) Cheyney (55) in 1924, stated that any patient with toxemia who develops retinitis has a four to one chance that she also has nephritis. He would only agree that occasionally there were cases in which an acute toxemia was present with no evidence of pre-existing nephritis and no evidence of nephritic residual. Masters (31) says that these patients

with toxemia and retinitis are definite candidates for permanent vascular kidney damage, the extent depending upon the time the retinitis is allowed to remain.

Vandegrift (31) states that the presence of sclerotic vessels in the fundus in a patient with toxemia denotes previous nephritis with hypertension, and the absence of such sclerosis denotes retinitis that is due to the pregnancy alone.

The lesions of the fundus usually disappear following delivery, unless the nephritis complicates the condition. (37) Some cases show marked visual loss due to transitory amaurosis caused by cerebral toxemia, but this returns to normal also. The changes seen in the retinal vessels are the underlying changes which cause all other lesions of the retina, and are only one of several pathological changes affecting arterioles of the body. Similar changes are generally considered to occur in the arterioles of the other organs of the body, such as in the glomeruli, the heart, and brain, and therefore, retinal evidences are simply reflections of a general process occurring throughout the arteriolar system. (64)

It is now quite generally believed and accepted that angiospasm frequently underlies these vascular pathological conditions. In fact some go so far as to say that retinal angiospasm is an almost constant accompanying factor of toxemias of pregnancy. (68)

In cases in which there is an associated rise in blood pressure, changes in the arterioles appear first, and retinal changes are secondary to and dependent upon these changes in the arterioles. It used to be thought that edema, exudates, and hemorrhages were the important factors, but now more attention is being paid to the retinal arteries. Mylius (55), in 1928, noted that the most common lesions of the fundus were spasms and tonic constrictions of the retinal arteries, and stated that the secondary retinal changes were due to the interference with the nutrition of the retina by these spasms.

As has been stated before, retinal angiospasm is the most common arteriolar finding in toxemia. These spasms may be intermittent or continuous, may affect small branches or segments of branches, or all of the branches of the retinal arterioles, and may show only a slight indentation of the vessels or a complete obliteration of the branches of the retina. All of the cases show this spasm, but the intermittent type of spasm seems to be the most frequently observed. (68) Bedell (28) states that the retinitis of pregnancy starts with an isolated spasm of one of the secondary or tertiary branches of the retinal artery, and then there is the development of the signs of true hypertension if the cause is not removed. Following this initial spasm there is an irregular constriction of the lumina of

of the arterioles, which varies and is usually more severe in the nasal branches of the retinal artery. This narrowing and constriction becomes more fixed and individual cotton-wool patches and hemorrhages appear in the retina, and finally there is a diffuse retinal involvement with exudates, hemorrhages, and the like.

Foster Moore (41) gives a different sequence of events in the development of the retinal lesions, and entirely ignores the vessel changes. Instead, he believes that the changes begin with the sudden onset of a tremendous exudation into the retina, which causes retinal detachment by the pouring of serum into the subretinal space. He also states that there is an equally sudden cessation of these findings upon removal of the cause, the pregnancy, if done promptly enough, and that there is a subsequent complete restoration of visual acuity.

However, the general idea among investigators is that already described concerning the development of spastic arteriolar retinal changes. Along with the arteriolar spasm, swelling of the nerve head is the most frequently noted initial sign. (55) The margins of the disc become indistinct, and are often reddened. The adjacent retina becomes edematous along with the nerve head as the edema is allowed to accumulate, and finally the entire fundus takes on a peculiar hazy

appearance due to the edema which has spread fairly generally throughout the retina. However, with all of this edema of the nerve head and the surrounding retina, it is generally considered that retinal detachment is an uncommon occurrence.

The veins of the retina, likewise, change, becoming very much engorged and enlarged in caliber in contrast to the smaller caliber of the arteries. As the engorgement progresses tortuosity and distortion occurs, and the fundus takes on the typical picture of hypertensive disease.

As time passes and the condition of the arterioles of the retina is allowed to remain, more permanent changes take place in the form of sclerosis. As these changes occur, the nutrition of the retina is disturbed and the changes secondary to the arteriolar changes begin to make their appearance. (12) White spots and patches in the stroma of the retina with the characteristic star-shaped figure in the macula appear just as in hypertension. These exudates are usually near the vessels, and even form the snow-bank masses which are seen to occupy a large part of the posterior pole of the fundus. Hemorrhages also occur, usually parallel and close to a large vessel, and usually being either flame-shaped or round, depending upon their depth in the tissue of the retina. (55)

In the toxemias of pregnancy the ophthalmoscope reaches its height as an instrument of prognosis and determining the future treatment of the patient as a whole. It is very uncommon to find retinal and optic nerve lesions in Pernicious Vomiting, but when such is found, Stander (31) has stated very definitely that there is an absolute indication for the immediate termination of the pregnancy. In the Low Reserve Kidney, Pre-Eclampsia, and Eclampsia types of toxemia, the retinitis is more marked, and retinal edema and detachment not so uncommon as before stated. In the Chronic Nephritis type of toxemia the retinal hemorrhages and renal type of retinitis predominates the picture. (41)

The problem of termination of pregnancy in toxic patients is a very difficult one to decide, and here is the place where the ophthalmoscope is of the greatest assistance to the obstetrician. Toxemia and its severity are still recognized by the usual signs of hypertension, albuminuria, edema and the symptoms presented, and the ophthalmoscopic picture fits in well with these other findings. (64) However, the repeated examination of the eyegrounds will not only show the extent of the pathological change, but may well indicate whether the process is stationary, advancing, or receding. It is here that the ophthalmoscope is the greatest aid. Early in pregnancy it permits the

detection of pre-existing renal and hypertensive disease, and later may reveal superimposition of new acute lesions on the old processes.

It is generally considered that when marked spastic changes occur in the retina early in pregnancy they indicate severe toxemic processes which endanger the viability of the child and result in future maternal health impairment. If these changes occur before the 28th. week of pregnancy, they lead to permanent cardio-renal-vascular disease in the mother. If they occur this early, only twenty five percent of the babies will be born alive. (68) Spastic changes later on in pregnancy are not a very grave prognostic sign. The presence of spastic changes which are fairly severe before the 28th. week of pregnancy is an indication for the termination of pregnancy and a contraindication for further pregnancies. (75)

Wagener (55) states that so long as the lesions in the arterioles are definitely spastic and the condition of the mother is otherwise satisfactory, it is safe to wait to terminate the pregnancy until after viability has been reached. Cotton-wool patches and retinal hemorrhages indicate the approach of permanent organic change, and pregnancy should be terminated to prevent the involvement of the renal arterioles and permanent kidney damage.

In cases in which there is rapid progression of

the fundus lesions, and increasing visual disturbance along with a marked general physical deterioration, it is usually advisable to terminate the pregnancy to save the patient's life and eyesight. (37)

The prognosis regarding vision in cases of retinitis is usually good. The prospects for the return of good vision are favorable unless there have been changes in the macula, retinal detachment, or a papilloretinitis. All of these conditions are fairly rare, and so perfect results as to the return of vision may be expected. (55)(31)

As one can easily see from this discussion of the topic of the retinitis of the toxemias of pregnancy, the ophthalmoscope is of tremendous value to the obstetrician, and to the general practitioner who does any obstetrics in his practice. However, even with its use, no rigid set of principles can be set up for the treatment of all cases in which retinitis is observed. Each case is an individual one, and must be treated as such, with the ophthalmoscope as a guide to when and in what manner the pregnancy must be ended to obtain the best results for mother and child.

XII. THE FUNDUS IN DISEASES OF THE BLOOD

1. Leukemia:

Leukemia is a disease characterized by a permanent increase in the leukocytes of the blood associated with hyperplasia of the leukopoietic tissues, and the occurrence of abnormal white cells in the blood stream. The etiology is quite obscure, the acute forms now being thought to be of an infectious nature, and the chronic forms on a neoplastic basis. (20)

The most common ocular manifestations of this disease are the fundus changes. Only about one-third of the cases reported have exhibited abnormal eyegrounds when examined. Borgeson and Wagener (38) in 138 cases, reported an incidence of seventy percent of fundus changes, but Moore points out that it is extremely doubtful that fundus changes are absent during the entire course of the disease in any patient. However, in Acute cases of Leukemia, the disease rarely lasts long enough for fundus changes to occur before the patient dies of the disease.

It is generally accepted that the changes which occur in the fundus occur in the periphery of the retina for the most part. (32) There are said to be three stages in the eyeground changes. (38)

The first stage is that in which there is only engorgement of the retinal veins noted. This represents

the earliest abnormality observed, and the most common one seen. The second stage is that in which in addition to the engorged veins, there is also an engorgement of the retinal arteries with a tendency for the arteries and veins to closely approximate each other in color. (23) Atkinson (3) disagrees with this finding of arterial engorgement, and states that instead there is a definite narrowing of the arteries, but does agree that there is marked similarity in the color of the veins and arteries. In this type there is also noted the presence of retinal hemorrhages and exudates. The third stage is that in which there is a diffuse pallor to the entire fundus, associated with papilledema and white streaks along the vessels, in addition to the changes already mentioned in the other two groups.

The paleness noted in the fundus seems to be quite a striking feature of the changes of the fundus. The entire retina is said to be very pale, and often gives a greenish-grayness to the entire fundus.(38) The veins and arteries are also included in this pallor, sometimes to such a great extent that they cannot be distinguished one from the other.

The hemorrhages in the retina of a leukemic patient may be of any type, but are the most frequently small and round with small white centers. These hemorrhages may be found in any part of the fundus, and as a rule

are fairly close to the vessels. They are said to be situated in the nerve fiber layer of the retina. The white centers have been proved upon pathological examination to be composed mainly of leukocytes. (38)

White deposits of exudates are quite commonly found in the retina of a leukemic patient. They usually cover a considerable area of the retina, and are at the same time located close to the vessels of the retina. (3) They consist of white flecks which have red borders, are small in size, irregular in shape, and are sometimes striated. (32) These areas are thought to arise due to the obstruction of the capillaries of the retina with white blood corpuscles, and are said by Buchanan to represent collections of leukocytes and degenerated nerve elements surrounded by traces of hemorrhages. (3) In some cases of leukemia there are very characteristic white streaks, as noted in stage three, which tend to follow closely the walls of both veins and arteries, and which differ from the exudative spots noted above on the retina. These streaks are considered to be caused by engorgement of the lymphatic vessels in the sheaths of the veins and arteries of the retina by white blood corpuscles. (3)

Involvement of the optic nerve head by edema and edema of the surrounding retina is rather an uncommon finding in this condition.

Visual disturbances may occur early or late in leukemia, but the typical fundus changes just described are found as a rule before there is any visual involvement. (32) This fact again stresses the importance of complete ophthalmoscopic examination of every patient.

2. Pernicious Anemia:

Pernicious anemia is a severe primary anemia caused by a deficiency in some substance or substances which are necessary for normal erythropoiesis. (20)

The most common ocular manifestation of pernicious anemia is the occurrence of retinal hemorrhages. In a review of 377 cases, Cabot (38) stated that retinal hemorrhages occurred in 72 percent of the cases. Some authorities believe that the presence of retinal hemorrhages depends upon the amount of hemoglobin in the blood, definitely stating that hemorrhages always occur with a hemoglobin level below thirty percent, and never occurring when the level is above thirty five percent. Others disagree with this, reporting that in their study of various series of cases there is no relationship between the occurrence of hemorrhages and the hemoglobin level of the blood.

The hemorrhages are usually small and scattered for the most part about the posterior pole of the fundus. They may be circular or flame-shaped, and at times have

white centers, very much similar to those of Leukemia. The hemorrhages rarely become large, and unless they are located in the macular region, there is very little likelihood that they interfere with vision. The hemorrhages which have white centers are called Roth's Spots, and are exactly the same as those seen in Leukemia. (38)

White spots occasionally are seen unassociated with hemorrhages in the retina. They are quite small, irregular in shape, and have hazy borders. They seem to be quite superficial in the retina, and may be located anywhere in the fundus. (23)

There is a lack of distention of the arteries of the retina, and an underfilling of the veins. This underfilling of the veins causes them to flatten out and appear enlarged, while the arteries appear smaller than normal. The entire fundus is also pale here as in Leukemia, but it is said to be a much more pronounced pallor than that seen in any other condition. (3)

The therapy of Pernicious anemia has become almost specific, and consists of the administration of liver extract. As the general disease progresses under this therapy, there is a gradual disappearance of the lesions of the retina, all parts of the lesions finally completely disappearing. (12)

3. Secondary Anemias:

The findings in the fundi of patients suffering from secondary anemias are quite similar to those of pernicious anemia, with the exception that there is considerable difference in the degree of involvement in the two. (3) In secondary anemias there is the pallor of the entire fundus, including the veins and arteries, but this is not nearly so marked as in pernicious anemia. The veins are also flattened out and appear enlarged, and the arteries are smaller than normal, and the pallor may be so marked here also that it is hard to distinguish arteries from veins. There may be a varying degree of retinal and nerve head edema present, too, but all of these signs quickly disappear as soon as the anemia undergoes adequate treatment. (8)

4. Following Hemorrhage:

There has been very little work done in the field of ophthalmoscopic examinations following blood loss, but what little work has been done definitely shows that fairly marked changes in the fundus do occur following severe hemorrhage. The most common finding is that of visual loss. (3) This is an infrequent complication when one considers the tremendous number of severe hemorrhages. However, it is present enough to warrant its consideration as one of the findings in

the fundus. It is said to have never been observed, however, following hemorrhage due to trauma, curious as this may seem. (38)

In most of the cases observed in which there was considerable and prolonged loss of blood there was a varying but definite swelling of the nerve head and adjacent retina, giving rise to stasis within the optic nerve, and a diminished blood supply to the retina. This accounted for the generalized narrowing of the vessels of the retina. (38) As this edema progresses there is usually the appearance of hemorrhages and exudates in the retina, and a pallor noted here as in the cases of the anemias. (12)

In a few cases there is the definite picture of an optic neuritis associated with retinal hemorrhages. Occasionally in a few weeks time, this neuritis picture will progress to the point where there is a post-neuritic optic atrophy, with generalized retinal thinning and degeneration, and in these cases the prognosis for the return of vision is very poor. (38)

5. Subacute Bacterial Endocarditis:

Subacute Bacterial Endocarditis is a disease in which there is an infection of the endocardium of the valves of the heart, usually by the streptococcus organism. These organisms form vegetations upon the

valve leaflets in the heart and may and frequently do break off, to be carried to various parts of the body by the blood stream as emboli.

The fundus manifestations of this disease are for the most part the result of the lodgement of these emboli in the end arteries of the retina. These emboli cause the appearance of petechiae in the retina, similar to those seen in the kidney, the brain, the conjunctiva, the skin, and other parts of the body. In the retina they give rise to hemorrhages which are small, round, and which have a yellowish-white nucleus or center. (38) This is the most common type of hemorrhage, but others have been noted to be flame-shaped, or perivascular in type. These hemorrhages may appear and disappear several times during the course of the illness, leaving no trace of their presence. They are generally considered to be quite a valuable diagnostic aid early in endocarditis. (51) All of these hemorrhages are not necessarily the result of emboli, some of them depending upon an associated anemia of the retina and general blood system for their development. (82)

Interest has been aroused of late to study the optic discs in these patients, because of the finding of marked papilledema in a patient having a normal spinal fluid pressure, but who had been confirmed to be a case of subacute bacterial endocarditis. Since the

discovery of this case a series of thirty seven cases have been studied and reported, and thirty five of these were found to have definite disc pathology. (50) There were several types of disc pathology noted, mainly optic neuritis, papilledema, embolic neuroretinitis, and combinations of these three. The factors which have been considered as pathogenic for the disc pathology include toxemia, embolic processes because of the evidence of emboli in the cerebral circulation shortly after or before the report of the fundus examination, and increased intracranial pressure, a factor which has been proved to have no bearing on this pathology. (46) There seemed to be a definite correlation between the nerve head pathology in these cases and visual disturbances which were found to occur. (51)

To give an idea of the incidence of fundus lesions which may be expected in cases of subacute bacterial endocarditis, eight cases were examined and the fundi of four found to be normal. One of the remaining four cases had choroidal scarring which was probably due to an embolus; one had a retinitis which had been caused by the anemia secondary to this disease; and two had embolic lesions such as have been described in the retina. Death in all eight cases was rather rapid in occurrence, and resulted from the septicemia present. (82)

A complication of Subacute Bacterial Endocarditis

which is not too uncommon is that of obstruction of the central artery of the retina by an embolus. (12) It causes sudden blindness, is unilateral in occurrence, and there is no pain accompanying it. The fundus becomes pale and edematous and takes on a milky appearance. The fovea becomes a very markedly red spot which stands out in contrast to the surrounding pale retina. The arteries are very thin and usually invisible a short distance from the nerve head. The veins are smaller than normal and may look beaded. After a few weeks atrophy of the retina occurs as well as atrophy of the nerve head which now shows up very white and sharply outlined. The arteries may be seen only as white streaks but some of the veins are seen to be full of blood.

XIII. THE FUNDUS IN SYPHILIS

Syphilis is a disease caused by the spirochete which has been called the great imitator because of its ability to imitate all other diseases in its manifestations. Among these manifestations are changes in the fundus of the eye which occur quite commonly. These changes usually involve both eyes, and are found with both acquired and hereditary forms of the disease. In the acquired form of syphilis the lesions of the fundus are found during the second stage of the disease, usually in the first or second year. In the hereditary form the lesions may be seen any time, and are not infrequently seen following interstitial keratitis. Luetic lesions occur in the eye early as well as late in the course of the disease. Mennesheimer has described lesions caused by syphilis as early as the fourth week after infection. (66)

The picture seen in the fundus of the eye with the ophthalmoscope in syphilis varies considerably, and is found to be quite different depending upon whether the disease is acquired or hereditary.

When the retina is involved alone by luetic infection which is acquired, there is usually first seen a clouding of the retina which is diffuse. This is due to swelling and edema of the retina, and to fine dust-like particles which accumulate in the posterior part

of the vitreous. These particles in some cases may be more marked in some places forming definite flecks. (73) The borders of the optic disc are somewhat blurred due to edema of the disc, and also due to the particles in the vitreous. There may also be a definite hyperemia of the nerve head, which if it persists for a long period of time may lead to optic atrophy. (3)

Changes in the retinal vessels in syphilis are quite prominent according to Roth. (66) These changes consist of sclerosis, the findings being practically the same as those previously discussed under the heading of arteriosclerosis.

The most predominant change in the fundus which is noted is that of pigmentary degeneration of the retina. This change usually starts with the appearance of small grayish or white spots in the retina which are bordered with pigment. These spots are the most marked in the region of the macula and in the periphery of the retina. As the condition progresses the pigment increases in amount until pigmentary degeneration of the retina occurs, usually accompanied by a similar degeneration in the choroid.

When the retina is involved alone there may be diffuse exudates on the retina accompanied by lines of exudation along the vessels. These may be quite broad and have a white fluffy appearance, or they may be more

or less elongated and feather-like. (3) Roth states that with the marked vesicular changes present which are due to sclerosis of the retinal vessels, the appearance of hemorrhages and exudates are quite characteristic. (66)

These changes just described are considered as typical of the type of ocular change known as Diffuse Retinitis Syphilitica. There is another type of change noted, which is not so diffuse but rather circumscribed, and which is generally considered to be localized near the macula. In this form there is a yellow or white exudate which forms and is quite extensive. It is seen as a rule in the macula or around one of the larger vessels. It is fairly well circumscribed, and is transformed later in the disease into a bluish-white scar, which may cause detachment of the retina by its shrinking. (12) After this scar is formed there is usually considerable pigment deposited in it.

Though involvement of the retina alone by the luetic infection is not an uncommon finding, the more common condition noted in the fundus is the involvement of the retina and choroid together. This condition is termed Syphilitic Chorioretinitis. When both are involved the first signs usually are dark spots scattered about in the fundus. These spots vary in number, but have a tendency to assume a round or oblong shape. The

borders of these spots are frayed, and many of them may coalesce at times to form larger areas. In these larger areas it is quite common to find small bare areas caused by depigmentation. (3)

There is a generalized cloudiness of the retina present which changes into patchy atrophy of the retina as the disease progresses. There are also numerous spots of choroidal atrophy present, and a large amount of pigment present, chiefly in the periphery of the retina.

In the hereditary form of syphilis there is usually both involvement of the retina and choroid. The changes present in the choroid resemble very closely retinitis pigmentosa, and are considered as specific for hereditary syphilis. (59) There are usually brownish discolorations of the fundus, with patches of pigment of various shapes and sizes which are present for the most part in the periphery of the fundus.

One case of hereditary syphilis was reported by Harry in the Canadian Medical Association Journal in 1938. This case was in a boy sixteen years of age who showed many stigmata of congenital syphilis. Upon ophthalmoscopic examination the right eye was seen to contain a large charcoal colored patch, which was irregular in contour, and bordered by a zone of sclerotic tissue. The disc was a chalky white, and the disc

arteries were absent. There were also two small patches of choroiditis with spots of pigmentation at 12 and 2 o'clock to the nerve head. The left eye showed much the same picture, except that the main charcoal lesion was in the region of the macula and extended downward, and that there was a more generalized pigmentation of the retina.

Occasionally there is seen in early cases of both tabes and paresis a low degree of optic atrophy which is secondary in character. There is usually a hyperemia of the nerve head for a long time previous to its development, and in some cases there is also a low grade papillitis present.(3)

Primary optic atrophy is the type which is the most commonly seen in syphilitics. It is such a common accompaniment of neurosyphilis, that its presence is considered to indicate lues until proved otherwise. The presence of primary optic atrophy in any patient should always demand very careful neurological and spinal fluid studies to rule out syphilis. This atrophy may become quite advanced and not disturb the patient's vision to any great extent. Any tabetic or paretic who complains of difficulty of any type with his vision should be suspected and examined for optic atrophy. (59)

The findings in the fundus which have just been discussed are the usual ones in syphilis. However,

occasionally other findings are noted which differ entirely from these. Such was the case in a case reported by Tshernoff.(73) In this case there was seen to be a mass the size of a nickel just below the macula of the left eye. This mass was conical in shape, and protruded into the vitreous. It was surrounded by a reddish exudate, and the retina over it and around it was pushed forward. The entire picture suggested malignancy of the eyeball, and it was generally considered that enucleation of the eyeball was the only hope. However, the physician decided to try a therapeutic test with Arsphenamine before surgery to completely rule out the chance of a luetic infection. Following a short period of treatment with Arsphenamine the mass began to become smaller, and eventually disappeared completely, with the vision and retina returning to normal. This case does not correspond to any generally accepted picture of syphilis of the retina or choroid, but shows how varied a picture one may see with a luetic infection.

XIV. THE FUNDUS IN TUBERCULOSIS

The ordinary type of pulmonary tuberculosis does not give the ocular picture to be described as frequently as does the miliary type, or tuberculous meningitis seen in children. However, even with the common everyday tuberculosis there are often signs in the fundus which may aid in the diagnosis. In these cases there is usually very little if any clinical evidence that there is tuberculosis in other parts of the body.

The involvement of the fundus of the eye by tuberculosis is mainly an involvement of the choroid, though secondary changes are seen to occur in the retina. The retina is not involved in all cases of tuberculous choroiditis, and is never seen to be involved alone. The changes in the choroid completely dominate the picture.

Manifestations of tuberculosis in the choroid usually show themselves as elevations, and as stated before are most commonly seen in miliary tuberculosis. These elevations appear as light yellowish or reddish-yellow spots in the choroid which are either round or oval. The edges of these elevations appear to be soft and fading, and are quite ill-defined. These areas are usually one to two millimeters in diameter, and are found most often near the disc. They are, however,

also found in the macular region and scattered over the periphery of the fundus in some cases. (3) The vessels in the area of elevation become partly or wholly obscured, and there are frequently small hemorrhages in or close to the area. (74) Microscopically these tubercles of the choroid are found to consist of giant cells and small round cells in which tubercle bacilli are found. (12)

Solitary lesions of the choroid are sometimes observed as elevations which are larger than those just described, and which have a yellow crest. They are always unilateral, and may cause a great degree of degeneration. Any tubercles of the choroid always involve the retina secondarily and cause some retinal depigmentation in the area involved. (3) Verhoeff (74) states that instead of this depigmentation there is a very definite accumulation of pigment in these areas, increasing as healing occurs, and leaving an atrophic irregularly pigmented scar in the choroid when the healing process is finished. The vitreous in these cases may become quite cloudy, and in some cases may even obscure the choroidal lesions from view.

Verhoeff (74) reported one case in which the findings in the fundus were exactly as stated above, and which upon pathological examination was proved to be tuberculous in origin. Previous to this report pictures in the

fundus such as described had been thought at times to be due to tuberculosis, but no proof had been obtained that they definitely were. With this pathological examination, Verhoeff states definitely that every fundus with this picture has tuberculosis.

One authority believes that there are two very definite findings in the eye which aid in the diagnosis of tuberculosis of the eye. These are first that there is relatively little pigment around the elevated lesion in the choroid, and second, that there is a curious light reflection almost like a phosphorescence seen coming from this lesion. (28)

Five cases of intraocular hemorrhages were reported, three of which were proved to be tuberculosis in origin. (70) In all of these three cases the complaint was that there was blurring of vision in one eye which had come on quite suddenly. In case one upon ophthalmoscopic examination the involved eye was found to have a massive hemorrhage into the vitreous which obscured the details of the fundus. In the other eye there were seen many small hemorrhages into the retina near the periphery of the fundus, with a slaty gray area of proliferation present in the retina. Old Tuberculin therapy was used in graduated doses, and the vitreous and retinal hemorrhages cleared up. However, they recurred again several years later and the patient had to undergo another series of

intramuscular injections of old tuberculin before they cleared up.

In the second case the unaffected eye showed a normal picture in the fundus. The affected eye showed a large elevated grayish mass in the macular region, and several patches of heavy, slaty-gray exudate in the periphery of the retina. There was also a large area of retinitis proliferans which extended into the vitreous. There were two rounded white lesions just below the nerve head which were considered to be choroidal tubercles. This case also cleared up almost completely with the use of Old Tuberculin therapy in small graduated doses.

The third case showed much the same findings as seen in the other two, and also definitely improved with the use of Tuberculin. From these cases it is definitely shown that the lesions of the fundus which are tuberculous in origin are usually benefitted and often entirely healed by Tuberculin therapy if it is properly given. (34)

Choroidal tubercles have been designated as the most conspicuous sign of ocular tuberculosis, while tubercles of the retina have been said to be extremely rare. However, there are nodules seen in the retina in some cases of tuberculosis which have frequently been called retinal tubercles, but which are instead areas of periphlebitis of the retina. The tubercles

of the choroid arise by spread of the infection by the blood stream. These retinal periphlebitic lesions of the retina are the result of a spread of the infection from a recent iridocyclitis. The extreme difficulty with which the retina is infected with the tubercle bacilli by the blood stream is shown in attempts to produce tuberculosis of the eye experimentally, such attempts failing in every case.

In addition to the nodules in the retina which are seen in Periphlebitis, there are areas of exudation around the veins, and profuse retinal hemorrhages and hemorrhages into the vitreous may occur. This condition often responds quite readily to cautious graduated doses of Old Tuberculin Intramuscularly.

XV. CONCLUSION

The ophthalmoscope is a diagnostic instrument used to examine the ocular fundus and its nervous and vascular elements. The changes noted in the retina, nerve head, and vasculature of the fundus are quite characteristic of the various general diseases discussed here. Because these changes are reflections of the general processes occurring in the other parts of the body, especially in the vital organs, the ophthalmoscope takes on additional importance. Not only is it of value in the diagnosis of local ocular disease, but also it now assumes a major role in the diagnosis of general systemic disease.

The ocular changes caused by disease are quite well known and easily recognized. The etiology and mechanism of production of these changes is still quite debatable and not well understood by the majority of authorities, though many theories have been advanced.

Finally, the value of the ophthalmoscope is greatly increased, because of the fact that the changes in the fundus can be observed carefully over a long period of time, and their progress noted. In this way the examiner knows what the outlook for the patient is, and when interference is indicated to improve this outlook.

To understand fully all of the changes discussed here as occurring in general disease one must make himself familiar with these pictures presented. For

this reason the inclusion of an ophthalmoscopic examination in every physical examination as a routine is strongly urged. Only in this way can one become expert in the use of the ophthalmoscope and appreciate its true value.

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